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location of tumor interact with regard to the HRQOL domains based on analysis of part of the cases.
CONCLUSION: While organ preservation is a major goal of surgical procedures, comprehensive, validated HRQOL surveys reveal new insights on the impact of sphincter-sparing vs. sphincter-ablating surgeries.

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P004
HISTORY OF URINARY TRACT INFECTIONS AND RISK OF RENAL CELL CARCINOMA
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PURPOSE: Limited data exist regarding whether a history of urinary tract infections (UTI) increases the risk of developing renal cell carcinoma (RCC). Further, there has been no effort to determine whether any association of RCC with a history of UTI is modified by known risk factors for RCC (i.e. smoking, obesity and hypertension). We report data from a population-based case-control study conducted in Iowa from 1986-1989.
METHODS: RCC cases (261 males; 145 females) were identified through the Iowa Cancer Registry, while controls (1,598 males; 831 females) were randomly selected from the general population, frequency matched on age and sex. Subjects provided detailed information on a mailed questionnaire regarding demographic, anthropometric, lifestyle, dietary and medical history risk factors, including self-reported history of a physician-diagnosed bladder or kidney infection.
RESULTS: In age-adjusted analysis there was clear evidence of an increase in risk for individuals who self-reported a history of physician-diagnosed infection of the kidney or bladder [Odds Ratio (OR) = 1.9, 95 percent Confidence Interval (CI): 1.5-2.5] compared with individuals reporting no such history. The risk of RCC associated with a history of UTI was more pronounced among males (OR = 2.7; 95% CI 1.9-3.8) and current smokers (OR = 4.3; 95% CI 2.7-6.7). No evidence of effect modification was noted with history of hypertension or BMI in the decade prior to RCC diagnosis. Overall, the strongest RCC risk was reported for male current smokers with a history of UTI (OR = 9.7; 95% CI 5.0-18.1). Multivariate adjustment for anthropometric, lifestyle and dietary factors associated with the development of RCC did not alter these findings.
CONCLUSION: The results of this study suggest a positive association of history of UTI and RCC development, with elevated risks most notable among males with a history of smoking.

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P005S
THE EFFICACY OF THE PERCENT FREE PROSTATE SPECIFIC ANTIGEN DENSITY IN SCREENING FOR PROSTATE CANCER IN PATIENTS WITH NORMAL DIGITAL RECTAL EXAMINATIONS AND INTERMEDIATE PROSTATE SPECIFIC ANTIGEN LEVELS
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PURPOSE: Patients with a normal digital rectal examination (DRE) and intermediate prostate specific antigen (PSA) level pose a significant problem for physicians since only 25% of these people have prostate cancer (PCa). Recently, percent free PSA density (%fPSAD) was developed in an attempt to increase the specificity of PSA testing for PCa in men with PSA concentrations between 4.1 and 10.0 ng/ml and benign findings on DRE. The purpose of this study was to determine if the %fPSAD would be an efficacious clinical test in screening for PCa compared to prostate specific antigen density (PSAD) and free-to-total prostate specific antigen ratio (%fPSA).
METHODS: A consecutive sample of 446 men, query of PCa, was referred for transrectal ultrasound (TRUS) and biopsy. Analysis of PSA and %fPSA were performed using Abbott Diagnostics kits (Abbott Laboratories, Canada). Of the 446 patients, 201 patients with intermediate PSA levels and benign DRE were selected from this sample. Statistical analysis to determine diagnostic utility for %fPSAD, PSAD and %fPSA included calculated sensitivity (Sens), specificity (Spec), and positive likelihood ratio (LR) with corresponding 95% confidence interval (CI) as well as Kappa statistic with an α level of $p = 0.05$.
RESULTS: Prevalence of PCa was 0.37+0.07 (74 $^{+}$PCa and 127 $^{-}$PCa). The %fPSAD (Sens = 96%; Spec = 28%; LR = 1.33) and %fPSA (Sens = 95%; Spec = 34%; LR = 1.44) tests demonstrated significant agreement ($p < 0.001$) with the biopsy test. However, PSAD (Sens = 95%; Spec = 10%; LR = 1.06), failed to demonstrate significant agreement ($p > 0.05$).
CONCLUSION: This study concluded that despite the agreement between %fPSAD and biopsy, the %fPSA demonstrated a higher level of specificity than %fPSAD.

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P006
BODY MASS INDEX, PHYSICAL ACTIVITY AND RISK OF COLON CANCER IN SHANGHAI, CHINA
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PURPOSE: Colon cancer incidence rates have been increasing rapidly for two decades in Shanghai, China, for reasons still unclear. High body mass index (BMI) and low physical activity (PA) are known risk factors for colon cancer in Western countries. We examined the joint effects of BMI and PA on colon cancer risk in a population-based case-control study in Shanghai
METHODS: In-person interviews were conducted with 931 patients newly diagnosed with colon cancer and 1552 randomly-selected controls aged 30-74 in Shanghai during 1990-1993. Information on height and weight history, leisure activities, mode of transportation, lifetime occupation, and other exposures, such as diet and lifestyle, were elicited. Body mass index was calculated as weight in kilograms (kg)/Square of the height in meters (m^2). Occupational physical activity (OPA) levels were derived from indices of sitting time and energy expenditure based on job history. Metabolic equivalents (METs) were estimated for various leisure (LPA) and transportation physical activities (TPA). Odds ratios

(ORs) and 95% confidence interval (CIs) were calculated to estimate colon cancer risk by anatomic subsites, using multiple logistic regression models. Interaction between PA and BMI was tested by adding an interaction term in the model.
RESULTS: Colon cancer risk increased significantly with usual BMI among men, with OR = 1.7 (CI = 1.1-2.4) among those in the highest quintile of BMI. The corresponding OR for women was 1.4 (CI = 1.0-2.1), with excess risk largely confined to pre-menopausal women. Risk was inversely associated with PA, regardless of the type of activities. Compared to those with high levels of TPA and OPA, persons with low levels of both activities had substantially elevated risk of colon cancer (OR = 2.8, CI = 1.3-4.1 for men, and OR = 3.9, CI = 1.6-5.1 for women). This association appeared to be more prominent for distal than proximal cancer. In both men and women, risk increased with low PA at each level of BMI, and with high BMI at each level of PA. Compared to those with lowest quintile of BMI and high PA, risk increased to nearly six fold in men (OR = 5.9, CI = 1.9-6.6) and threefold in women (OR = 2.8, CI = 1.6-5.1) with highest quintile of BMI and low PA.
CONCLUSION: High BMI and low PA increased the risk of colon cancer even in the relatively low-risk Chinese population. Risk was further increased with significant effect modification between these two risk factors.

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P007
USING KNOWLEDGE OF DEVELOPMENTAL BIOLOGY TO MINIMIZE CONFOUNDING IN THE STUDY OF MENOPAUSE AND MENINGIOMAS: A POPULATION-BASED STUDY

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PURPOSE: To investigate the relationship between menopause and meningioma risk, we utilized knowledge from developmental biology to stratify risk by anatomical location. Results from the Mifepristine clinical trial question the validity of the hypothesis that ovarian hormones promote the development of meningiomas in women. Moreover, recent discoveries in embryology suggest that the meningeal membranes in the calvarium and skull base are regulated by independent developmental pathways.
METHODS: We evaluated the correlation between age at diagnosis and incidence of meningiomas, using population-based data from the Central Brain Tumor Registry of the United States (CBTRUS). We stratified our results by anatomical location to reflect different developmental pathways regulating embryogenesis, thus minimizing confounding.
RESULTS: Using Linear Regression models and Joint-Point analysis, we found that expected menopausal age is protective for tumors arising in the cerebral hemispheres (the second-degree polynomial T ratio was statistically significantly better fit than the linear model, Wald test $p = 0.0006$), but not in the skullbase (there was no difference between the linear, first and second degree polynomial, Wald Test $p > 0.05$). Paradoxically, female preponderance is much greater in the skullbase.
CONCLUSION: The lack of a protective effect of menopause in skullbase tumors suggests another gender-dependent mechanism of action. This result indicates the plausibility of unrelated oncogenic pathways leading to the development of meningiomas. The traditional ovarian hormonal hypothesis must be carefully reevaluated in future research, however our analysis supports this hypothesis only for calvarium meningiomas. We conclude that the etiology of meningiomas is contingent upon tumor location. Future studies investigating the molecular and cellular mechanisms of meningioma development must take these factors into consideration.

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P008
MENOPAUSAL ESTROGEN THERAPY AND ENDOMETRIAL CANCER IN A US COHORT: RECENCY AND POTENTIAL INTERACTIONS WITH OTHER RISK FACTORS

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PURPOSE: Menopausal estrogen therapy (ET) increases endometrial cancer risk, but certain key aspects of this association are unknown, including whether risk disappears after ET cessation and whether risk is higher in women with lower body mass index (BMI) or in smokers.
METHODS: The NCI's Breast Cancer Detection Demonstration Project Follow-up study included 61,431 women. To analyze data for the 30,509 women who reported a natural menopause and were at risk of endometrial cancer, we excluded women who reported menopause due to surgery, radiation, or unknown reasons, and women who developed endometrial cancer, had a hysterectomy, or died before baseline. We collected ET and other data at up to 6 telephone interviews and 3 mailed questionnaires between 1979 and 1998. Poisson regression with time-dependent variables for ET and smoking produced rate ratios (RRs) with 95 per cent confidence intervals (CIs), adjusted for other known endometrial cancer risk factors.
RESULTS: We identified 541 endometrial cancers but limited this analysis to person-years and cancers among users of no hormone therapy (168 cancers) or only ET (167 cancers). Risk increased with increasing ET duration, and RRs decreased with increasing time since last use, from 10.5 (95% CI, 7.5-14.9) for current use to 1.6 (95% CI, 1.1-2.3) for last use 10 or more years ago. Elevated RRs appeared for long-term ET, regardless of recency, and for current ET, regardless of duration. The RRs for 5 or more years of ET declined across increasing quartiles of BMI, from 13.0 among $< = 21.3$ kg/m^2, to 16.0 among 21.3-23.2 kg/m^2, to 7.4 among 23.2-25.8 kg/m^2, to 2.4 among $> = 25.8$ kg/m^2. Current smokers had a significantly reduced RR, but RRs for 5 or more years of ET among current, former, and never smokers were 28.5, 4.3, and 6.8, respectively; all CIs excluded 1.0.
CONCLUSION: Endometrial cancer risk decreased with increasing time since last ET use, but remained significantly elevated even 10 years after last use. Lower BMI and current smoking may exacerbate risk associated with ET, but significantly increased risks were not limited to these groups.

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